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Lewis C. Cantley, PhD, and José Baselga, MD, PhD, Editors-in-Chief

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ONLINE For more News and Research Watch, visit *Cancer Discovery* online at www.AACR.org/CDnews.

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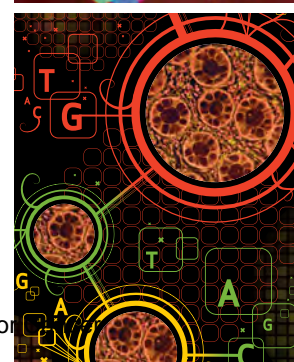
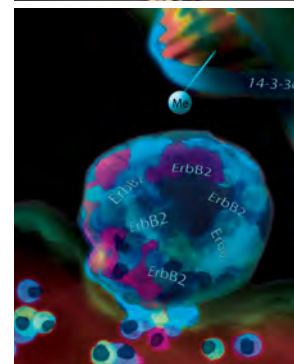
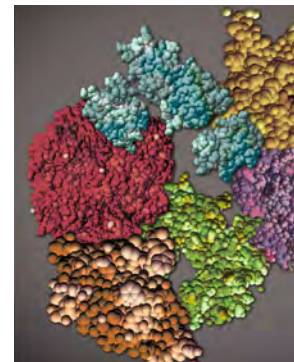
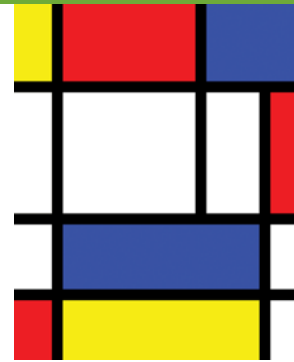
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A. DeCensi, B.K. Dunn, M. Puntoni, A. Gennari, and L.G. Ford

RESEARCH BRIEFS ATM Mutations in Patients with Hereditary Pancreatic Cancer 41
N.J. Roberts, Y. Jiao, J. Yu, L. Kopelovich, G.M. Petersen, M.L. Bondy, S. Gallinger, A.G. Schwartz, S. Syngal, M.L. Cote, J. Axilbund, R. Schulick, S.Z. Ali, J.R. Eshleman, V.E. Velculescu, M. Goggins, B. Vogelstein, N. Papadopoulos, R.H. Hruban, K.W. Kinzler, and A.P. Klein

Précis: Next-generation sequencing identifies inherited ATM mutations in kindreds with hereditary pancreatic ductal adenocarcinoma.

Molecular Ontogeny of Donor-Derived Follicular Lymphomas Occurring after Hematopoietic Cell Transplantation 47
 *O. Weigert, N. Kopp, A.A. Lane, A. Yoda, S.E. Dahlberg, D. Neuberg, A.Y. Bahar, B. Chapuy, J.L. Kutok, J.A. Longtine, F.C. Kuo, T. Haley, M. Salois, T.J. Sullivan, D.C. Fisher, E.A. Fox, S.J. Rodig, J.H. Antin, and D.M. Weinstock*

Précis: Analysis of a donor-recipient pair with follicular lymphoma reveals the time-course of somatic mutations acquired during lymphomagenesis.



**RESEARCH
ARTICLES**

**Genomic Complexity and AKT Dependence
in Serous Ovarian Cancer 56**

*A.J. Hanrahan, N. Schultz, M.L. Westfal, R.A. Sakr,
D.D. Giri, S. Scarperi, M. Janikariman, N. Olvera,
E.V. Stevens, Q-B. She, C. Aghajanian, T.A. King,
E. de Stanchina, D.R. Spriggs, A. Heguy, B.S. Taylor,
C. Sander, N. Rosen, D.A. Levine, and D.B. Solit*

Précis: Individualized analyses of the PI3K/AKT and RAS pathways will identify ovarian cancers that may respond to AKT inhibition.

**Loss of the 14-3-3 σ Tumor Suppressor
Is a Critical Event in ErbB2-Mediated
Tumor Progression 68**

C. Ling, V-M-T. Su, D. Zuo, and W.J. Muller

Précis: 14-3-3 σ inactivation accelerates formation and promotes metastasis of ErbB2/HER2-induced tumors.

**High-Throughput Detection of
Actionable Genomic Alterations
in Clinical Tumor Samples by
Targeted, Massively Parallel
Sequencing 82**

*N. Wagle, M.F. Berger, M.J. Davis,
B. Blumenstiel, M. DeFelice, P. Pochanard,
M. Ducar, P. Van Hummelen, L.E. MacConaill,
W. C. Hahn, M. Meyerson, S.B. Gabriel, and
L.A. Garraway*

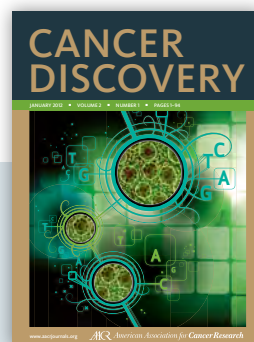
Précis: Targeted, sequencing-based profiling of archival tumor samples identifies genetic alterations that can direct personalized therapy.

For more News and Research Watch, visit *Cancer Discovery* online at www.AACR.org/CDnews. Online-only News stories include the following:

- Biotech Firms Look for Virtual Success
- Dual HER2 Blockade Slows Metastatic Breast Cancer
- “Reversed” Krebs Cycle Can Feed Tumors
- Modified Stem Cells Create Tumor-Attacking T Cells

**ON THE
COVER**

Wagle and colleagues describe a method to profile clinically relevant mutations in formalin-fixed, paraffin-embedded tumor samples involving exon capture of frequently mutated or polymorphic genes followed by massively parallel sequencing. This method identifies single-nucleotide variants, insertions, deletions, and copy number alterations overlooked by current genotyping-based methods with high specificity and sensitivity. Identification of such “actionable” genetic alterations that predict response to targeted or conventional cytotoxic therapies has the potential to facilitate individualized cancer treatment in a time- and cost-effective manner. For details, please see the article by Wagle and colleagues on page 82.



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